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AML PRESENTING AS ACS: A CASE REPORT

Medicine

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ABSTRACT

Acute coronary syndrome (ACS) commonly result from atherosclerotic occlusion of coronary arteries but rarely coronary spasm, substance abuse, systemic vasculitis and coronary embolism can cause the ACS with normal coronary arteries. Acute myeloid leukemia (AML) has been reported as a rare cause of ACS, here we report a 50 year old male, severely anemic, non-diabetic, non-hypertensive, alcoholic and smoker who presented with severe retrosternal chest pain. Search for secondary cause of ACS in view of severe anemia revealed him to be suffering from AML. He was treated for ACS and chemotherapy was planned for AML.

KEYWORDS

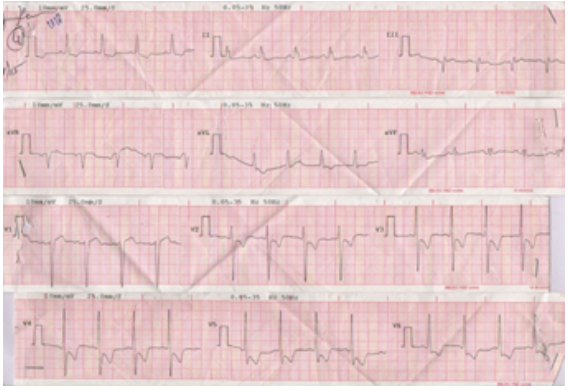
1. Bicytopenia 2.Pancytopenia 3.Coronary Spasm 4.Myocardial Infarction 5. Leukemic Blasts

CASE REPORT

A 50 Year old male, presented with severe retrosternal chest pain associated with diaphoresis which awakened the patient from sleep early in the morning. No complaint of dyspnea, palpitation and vomiting. Past history did not reveal evidence of bleeding tendency, fever, night sweats or weight loss. Patient was non diabetic, non-hypertensive, alcoholic and chronic smoker for 20 years. On examination patient was pale looking severely anemic, no evidence of bleeding tendencies or vitamin deficiency, pulse rate 80/min, BP 110/70 mm of Hg, respiratory rate 18/min and Jugular Venous pulse was not raised. Cardiovascular system examination revealed apex beat in 5thleft Intercostal space with both S1, S2 normal, no added sounds or murmur. Abdominal examination did not reveal any organomegaly. Respiratory and Neurological examination was normal.

Investigations revealed Hemoglobin of 4.2gm/dl, normal total leukocyte count of 7000/cumm and reduced platelet count of 19000/cumm. Peripheral smear showed atypical cells. Renal and Liver function tests were normal.

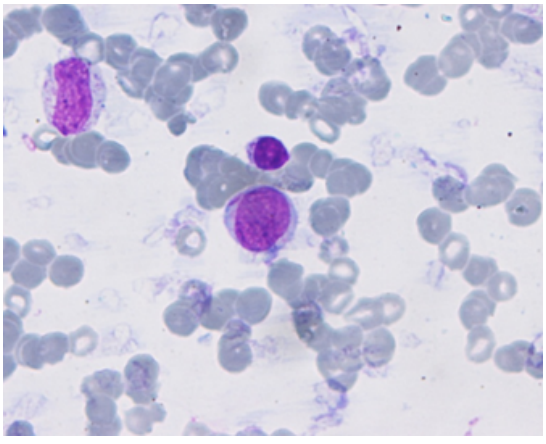
Electrocardiogram at presentation showed marked ST Segment depression and T wave inversion in lead V2-V6 and T wave inversion in lead 1 and avL suggesting lateral and anterior wall ischemia [Table/Fig 1]. Transthoracic echocardiography revealed hypokinesia of basal inferior and mid inferior wall with Left Ventricular Ejection Fraction 55%. Cardiac biomarkers CPK-MB and troponin-T were elevated. CT angiography revealed normal coronary arteries.



[TABLE/FIG 1]: ECG showing Inferior and Anterior wall ischemia

In view of his bicytopenia and atypical cells on PBF, bone marrow biopsy was done and it revealed AML M2 subtype [Tble/Fig 2]. Further cytogenetic studies could not be done.

The diagnosis of ACS with AML was made. The patient was treated on the lines of ACS with anti-ischemic and atorvastatin 80mg and 2 units of PRBC were infused, his chest pain subsided and patient was planned for AML chemotherapy. Patient is being followed up and he is on chemotherapy for the same.



[TABLE/FIG 2]: AML M2 (showing Auer Rods) Wright-Giemsa staining (magnification, × 1,000)

DISCUSSION

Acute Coronary Syndrome(ACS) most commonly result from atherosclerotic occlusion of coronary arteries but rarely coronary spasm, substance abuse, systemic vasculitis and coronary embolism can cause the ACS with normal coronary arteries. Acute Myeloid Leukemia generally present as shortness of breath, easy bruising, weakness, increased risk of infections and bleeding. ACS as an initial presentation of acute leukemia has been reported very rarely[1,2,3]. Our patient has AML M2 sub type resulting in severe anemia which lead to ACS secondary to ischemic imbalance, type II Acute Myocardial Infarction(AMI). Myocardial infarction can be spontaneous due to atherosclerotic plaque rupture (type1), secondary to an ischemic imbalance (type2), cardiac death due to MI without available biomarkers value (type3), associated with Percutaneous Coronary Intervention(PCI) (type4), stent thrombosis and Coronary Artery Bypass Graft(type5)[4]. AML can cause cardiovascular diseases either by leukemic cell infiltration into the myocardium or pericardium, occlusion of coronary arteries by leukemic thrombus, effect of anti leukemic therapy such as chemotherapy or radiotherapy resulting in leukostasis due to increased adhesivity, decreased deformability of the leukemic blasts, coagulation disorder caused by leukemic process, disseminated intravascular coagulation, hyperfib rinolysis or deficiency of coagulation factors [1].

In treating AML, induction therapy eliminates blast cells resulting in survival benefits but chemotherapy induced thrombocytopenia leads to bleeding tendencies, to prevent this, a platelet transfusion is recommended to achieve a target platelet count of 50 to 100 × 109/L5,6.

Despite thrombocytopenia in AML, platelet-fibrin thrombus may form in a major epicardial coronary arteries for which instead of Dual Anti

Platelet Therapy and anti-coagulant, balloon angioplasty with provisional use of bare metal stents is recommended to prevent major bleeding. Another useful intervention is PCI via radial approach that reduces the risk of bleeding complications[1,7,8].

The prognosis in concomitant STEMI and AML is clearly worse than that of either pathologic condition individually.

CONCLUSION: AML generally present as shortness of breath, easy bruising, weakness, increased risk of infections and bleeding, scanty reports of AML present as ACS. It seems useful to sensitize all clinicians to this complication of AML by reporting a case observed in our institution.

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